

Brief Reports

Influence of Personality Disorders on Therapy Outcome in Somatoform Disorders at 2-Year Follow-up

Comorbidity of personality disorders (PD) in axis-I disorders was found to be a negative predictor for treatment outcome in most studies up to the early 1990s (Reich and Vasile, 1993). However, recent studies on cognitive-behavioral treatment (CBT) report no influence of PDs on therapy for depression (Hardy et al., 1995; Shea et al., 1990), anxiety (Dreessen et al., 1994), and obsession-compulsion (Dreessen et al., 1997). Conflicting results might be explained by differences in assessment methods (*i.e.*, development of standardized clinical interviews and evolution of diagnostic criteria), size and composition of patient samples, and treatment strategies (medical treatment *vs.* psychotherapy). Results of Hardy et al. (1995) and Shea et al. (1990) revealed that comorbid PDs negatively affected the therapy outcome of interpersonal therapy but not of CBT. Severity of axis II pathology might be another important factor determining effects on therapy outcome of axis I disorders. In DSM-IV (American Psychiatric Association, 1994), PDs are classified by three clusters. Cluster C (anxious) PDs are commonly regarded as less severe than cluster B (dramatic) and cluster A (eccentric) PDs, which is addressed insufficiently in recent studies investigating the influence of axis II comorbidity on CBT treatment outcome of axis I disorders. PDs of cluster A and B might affect therapy more than less severe PDs of cluster C.

Little is known about comorbidity of PDs and somatoform disorders (SFD). Rost et al. (1992) found a frequency of about 50% PDs in SFDs, Stern et al. (1993) report a rate of about 70%. There has been no substantial evidence about the influence of PDs on treatment outcome of somatoform disorders until now. Treatment of SFDs, in particular somatization disorder, is regarded as difficult and ineffective (Barsky, 1988). However, recent findings suggest that a clear and stable improvement of somatoform symptoms may be attained by CBT programs in primary care patients as well as in inpatients with severe and chronic SFDs (Rief et al., 1995; Speckens et al., 1995).

The aim of the present study was to determine the significance of different types of PDs for therapy outcome of SFDs. We focused on three different aspects of this question. First, we analyzed differences between somatoform patients with and without comorbid PDs on pathology indices at the beginning of treatment. Second, we evaluated treatment outcome of the total sample to obtain more information about the effectiveness of our SFD therapy program in general. Third, we investigated differences in treatment outcome between the six subgroups to analyze the influence of comorbid PDs.

Methods

Subjects: We investigated a sample of 120 inpatients consecutively admitted to the Klinik Roseneck Center for Beha-

vioral Medicine in Prien, Germany, which specializes on the predominantly psychotherapeutic treatment of patients suffering from multiple and chronic mental disorders that are too severe for outpatient treatment. Patients with schizophrenia, primary eating disorders, and primary alcohol/substance use disorders were excluded from the study. All patients obtained the CBT program offered in our clinic. It consisted of individual therapy (completed by guidelines for the treatment of SFD) and group therapy sessions as well as standardized therapy programs focusing on specific parts of the psychopathology (e.g., pain, depression, or anxiety), social skills training, progressive muscle relaxation, and further psycho and physio-therapeutic treatments. All subjects participated voluntarily in this study after informed consent was obtained.

Assessments: Patients completed a battery of self-rating scales at least 4 weeks before treatment, at the beginning and the end of treatment, and 2 years after discharge. Somatoform symptoms were measured by the Screening for Somatization Symptoms (SOMS-7; Rief et al., 1997), a 53-item self-rating scale including all somatoform complaints listed in DSM-IV and ICD-10. The patients also completed the Whiteley Index (Pilowsky, 1967), a 14-item scale to measure hypochondriasis, the Beck Depression Inventory (BDI; Beck and Steer, 1987), and a recently developed 48-item self-rating scale, the Cognitions About Body and Health Questionnaire (CABAH; Rief et al., 1998) measuring dysfunctional cognitions about body and health on the five subscales Catastrophizing Cognitions, Intolerance of Bodily Complaints, Bodily Weakness, Autonomic Sensations, and Health Habits. Diagnostic interviews were guided by the International Diagnostic Checklists for DSM-IV (IDCL; Hiller et al., 1996) and IDCL P (Bronisch et al., 1996) for personality disorders, which are recommended by the WHO to obtain reliable and valid diagnoses. To control for severe depression and effects of acute symptomatology on the validity of personality disorder diagnoses (Zimmerman, 1994), the diagnostic interviews of axis II were not conducted until 4 weeks after admission to treatment, and depressive symptomatology was measured by the Depression Status Inventory (DSI [expert version]; Zung, 1972). One subject who scored more than 60 in the DSI (which is the proposed cut-off for medium-sized to severe depression) was excluded from the study.

Definitions: In addition to the somatoform disorder categories provided by DSM-IV, the Somatic Symptom Index (SSI 4/6; Escobar and Canino, 1989), comprising at least four somatoform symptoms for men and six for women, was applied to cover clinically relevant somatoform syndromes below the threshold of somatization disorder. The SSI-4/6 concept was developed as an abridged somatization construct, because somatization disorder according to DSM-IV criteria is very rare with a prevalence of less than 1% in the community,

whereas lifetime prevalence of SSI-4/6 is 4.4% (Escobar and Canino, 1989). Undifferentiated somatoform disorders and somatoform disorders NOS were excluded from the study.

Group Definition: We decided to make no subclassification of SFDs because the focus of this study was to compare different types of comorbidity. The total sample was classified into somatoform patients with/without PDs and nonsomatoform controls with/without PDs. Somatoform subjects with comorbid PDs were subclassified again along dimensions of severity of the axis II pathology: somatoform patients with cluster A and B diagnoses (both characterized by higher levels of impairment) were separated from patients with cluster C diagnoses, and single/multiple diagnoses of cluster C were differentiated because multiple diagnoses (despite a certain overlap of criteria) might indicate a more generalized axis II pathology than single PD diagnoses. Subclassification procedure resulted in six subgroups: a) SFD without PD (SFD), b) SFD and 1 cluster C diagnosis (SFD + PD-C), c) SFD and multiple cluster C diagnoses (SFD + PD-CC), d) SFD and cluster A or B diagnoses (SFD + PD-A/B), e) nonsomatoform controls without PD (CONTR), and f) nonsomatoform controls with PD (CONTR + PD).

Statistical Analyses: Differences between the six groups at any assessment were analyzed by one-way analyses of variance and Scheffé-corrected post-hoc comparisons. Treatment effects and global differences between the groups regarding therapy outcome were evaluated by MANOVAs with repeated measures (4 assessments), and effect sizes (difference of means of assessments T0/T1 and T3 divided by the pooled standard deviation of the entire sample in T0/T1) for each group were calculated to compare improvements directly. *t*-Tests for the improvement of each group on every scale were not conducted because validity of significance levels is a concern in 48 tests.

Results

The Sample: Of 119 subjects entered in the study, 97 (82%) completed all questionnaires. Drop-out rate was significantly higher in patients with comorbid PDs than in those without PD (26.9% vs. 7.7%; $\chi^2 = 5.9$; $p < .01$). We found no significant differences in age (mean = 44.8; $F = 1.0$; NS), gender (65% female; $\chi^2 = 3.5$; NS), marital status (67% married; $\chi^2 = 2.4$; NS), or treatment duration (mean = 61.2 days; $F = .9$; NS) between the six subgroups. Overall differences in educational level (1 = low to 8 = high) were significantly different (mean = 4.2; $F = 2.4$; $p < .5$), but no significant differences between two subgroups occurred in the single comparisons. In the diagnostic interviews of axis I and II, 95 patients met criteria for at least one somatoform disorder. Frequencies for specific diagnoses were: 28 (26.7%) somatization disorder, 58 (55.2%) SSI 4/6, 44 (41.9%) pain disorder, 31 (29.5%) hypochondriasis, 13 (12.4%) conversion disorder, and 2 (1.9%) body dysmorphic disorder. Sixty-seven patients (56.3%) met criteria for at least one personality disorder (PD); the following frequencies were observed: 39 (32.8%)

avoidant PD, 33 (27.7%) obsessive-compulsive PD, 13 (10.9%) dependent PD, 10 (8.4%) narcissistic PD, 8 (6.7%) histrionic PD, 5 (4.2%) borderline PD, 3 (2.5%) schizoid PD, and 3 (2.5%) paranoid PD. Axis I comorbidity rate was 79.0% for major depression and 68.9% for anxiety disorders.

Group Differences at the Beginning of Treatment: At the beginning of treatment a significant overall difference between the six subgroups was found only for the depression score (BDI), but no differences between two subgroups occurred in the Scheffé-tests (Table 1).

Group Differences in Treatment Outcome: We found significant global improvements of pathology on seven of eight scales applied, whereas no significant interaction effects occurred in the repeated measures MANOVA, indicating that treatment outcome of the six subgroups was not clearly distinct. However, the results of the effect size analysis suggest a slightly poorer outcome of SFDs with more severe types of axis II comorbidity. Subjects with SFDs and multiple cluster C PDs as well as those with SFDs and cluster A/B PDs did not improve on the SOMS-7 score. Outcome of somatoform symptoms in SFD patients with multiple cluster C PDs was even negative. Regarding hypochondriacal symptoms and the CABAH subscale Intolerance of Bodily Complaints, improvement was only moderate in this group. Moreover, SFD patients with multiple cluster C PDs are the only SFD group with significantly elevated levels of somatoform symptomatology compared with the control group without PD at 2-year follow-up. However, treatment outcome of SFD subjects with multiple cluster C PDs or cluster A/B PDs is not worse regarding the depression score and 4 of 5 CABAH subscales (Table 1).

Discussion

The fact that we found no differences in clinical characteristics (such as depression or hypochondriasis) between SFD subjects with and without comorbid PDs is somewhat unexpected because former studies reported elevated levels of psychopathology in subjects with comorbid PDs (Hardy et al., 1995; Shea et al., 1990). However, a general characteristic of our sample is that comorbidity of various disorders of axis I and II (especially depression, anxiety, and PD) is very large. For this reason, high levels of psychopathology on the measures applied in the present study are plausible for all groups investigated. On the other hand, PDs of cluster A or B in our sample might be mild varieties, whereas severe types of these disorders are more likely to be discovered in psychotic patients or on forensic wards.

Our results suggest that a significant and durable improvement of psychopathology may be attained in somatoform patients even when severe types of axis II comorbidity are present. However, treatment outcome is only moderate with respect to somatoform symptomatology, whereas better effects were found for depression, hypochondriasis, and cognitions about body and health. One reason might be that our treatment program included guidelines for the individual therapy but no specific standardized treatment program for somatoform symptoms, which are very persistent. Regarding

TABLE 1
Group Differences and Treatment Outcome

Entire sample	Somatoform patients						(Non-somatoform) Controls						ANOVA		MANOVA			
	I cluster C PD			I multiple Cluster C BPD			I at least 1 Cluster A or PD			Without comorbid PD			With any comorbid PD			Overall differences between groups	Significant single comparisons	
	I	II	III	III	III	III	IV	V	VI	V	VI	V	VI	WS effect				WS effect
(N = 95)	(N = 35)	(N = 16)	(N = 12)	(N = 13)	(N = 11)	(N = 8)	(N = 11)	(N = 8)	(N = 11)	(N = 8)	(N = 11)	(N = 8)	(N = 11)	(N = 8)	(N = 11)	(N = 8)		
SOMS-7																		
T ₁	32.4 (20.4)	36.6 (21.3)	33.9 (22.8)	39.3 (20.4)	30.6 (17.3)	18.9 (14.0)	2.8 (16.4)											
T ₂	22.9 (18.3)	25.1 (18.4)	22.2 (19.8)	31.0 (14.8)	28.4 (20.3)	8.7 (8.8)	12.6 (14.5)											
T ₃	28.9 (26.3)	31.1 (25.8)	27.4 (23.5)	48.0 (38.7)	31.5 (22.6)	10.5 (9.6)	15.0 (11.6)											
Effect size T ₀ T ₁	0.2	0.3	0.3	-0.4	0.0	0.4	0.4											
Whiteley-Index																		
T ₀	(N = 89)	(N = 32)	(N = 15)	(N = 11)	(N = 12)	(N = 12)	(N = 7)											
T ₁	6.4 (3.4)	6.9 (3.2)	7.5 (3.2)	6.5 (4.5)	7.1 (3.2)	3.8 (2.4)	5.1 (2.9)											
T ₂	6.1 (3.2)	6.5 (3.0)	6.5 (3.0)	6.8 (3.8)	6.2 (3.8)	4.5 (2.6)	4.3 (3.7)											
T ₃	4.1 (3.5)	4.7 (3.3)	4.0 (3.9)	5.5 (5.2)	3.9 (3.0)	3.0 (2.3)	1.4 (1.1)											
Effect size T ₀ T ₁	0.4	0.4	0.3	0.2	0.5	0.3	0.2											
BDI	(N = 89)	(N = 35)	(N = 15)	(N = 11)	(N = 10)	(N = 10)	(N = 8)											
T ₀	21.3 (10.1)	20.7 (9.6)	19.1 (9.9)	28.0 (11.5)	23.7 (11.5)	21.0 (7.5)	16.8 (10.2)											
T ₁	20.6 (10.3)	20.2 (9.4)	18.0 (10.2)	29.9 (9.7)	22.3 (11.2)	18.0 (8.8)	15.7 (10.1)											
T ₂	10.3 (9.4)	11.2 (8.5)	7.1 (6.4)	15.3 (10.9)	14.1 (13.7)	6.6 (7.9)	5.3 (7.0)											
T ₃	14.1 (10.8)	16.1 (9.9)	11.4 (9.6)	21.1 (12.7)	15.4 (12.8)	7.4 (8.6)	7.1 (5.2)											
Effect size T ₀ T ₁	0.7	0.5	0.8	0.7	0.8	1.3	1.0											
CABAH 1	(N = 92)	(N = 35)	(N = 15)	(N = 11)	(N = 12)	(N = 11)	(N = 8)											
T ₀	20.3 (11.1)	21.3 (9.3)	22.4 (11.7)	21.8 (13.9)	20.9 (15.6)	18.8 (9.0)	11.1 (4.1)											
T ₁	19.9 (10.4)	21.3 (8.6)	22.1 (11.6)	22.4 (13.1)	19.9 (12.7)	17.9 (8.6)	9.5 (4.4)											
T ₂	16.5 (10.6)	18.5 (9.8)	16.0 (9.6)	20.3 (13.4)	16.3 (13.6)	13.0 (9.6)	8.8 (4.1)											
T ₃	16.8 (10.1)	18.9 (8.8)	16.4 (11.5)	18.3 (13.6)	17.4 (11.6)	14.1 (7.4)	8.9 (10.1)											
Effect size T ₀ T ₁	0.3	0.2	0.5	0.3	0.3	0.4	0.2											
CABAH 2	(N = 82)	(N = 31)	(N = 14)	(N = 10)	(N = 11)	(N = 9)	(N = 7)											
T ₀	6.7 (3.8)	7.4 (3.7)	6.9 (3.5)	7.2 (4.8)	7.1 (4.7)	4.3 (2.4)	4.4 (1.7)											
T ₁	6.6 (3.6)	7.2 (3.4)	7.1 (2.8)	7.6 (5.0)	7.3 (4.4)	4.2 (2.1)	3.9 (2.4)											
T ₂	5.5 (3.8)	6.1 (3.7)	5.4 (3.0)	7.1 (5.3)	6.1 (4.4)	2.6 (2.1)	3.4 (1.1)											
T ₃	5.8 (3.7)	6.4 (3.9)	4.1 (2.0)	7.4 (4.8)	6.5 (4.3)	3.2 (2.6)	6.0 (2.1)											
Effect size T ₀ T ₁	0.2	0.3	0.7	0.1	0.2	0.3	0.1											
CABAH 3	(N = 85)	(N = 31)	(N = 16)	(N = 11)	(N = 11)	(N = 8)	(N = 8)											
T ₀	13.0 (4.9)	13.4 (4.5)	11.7 (5.0)	16.3 (5.4)	12.0 (3.2)	11.5 (7.3)	12.3 (3.6)											
T ₁	12.8 (4.7)	12.8 (3.9)	12.4 (5.3)	16.1 (5.4)	11.5 (4.7)	12.9 (5.5)	10.8 (3.7)											
T ₂	11.1 (5.4)	11.7 (4.1)	8.6 (4.7)	15.6 (5.6)	11.9 (5.9)	10.3 (6.4)	7.0 (3.5)											
T ₃	11.6 (5.7)	12.3 (5.2)	9.8 (6.1)	15.1 (5.2)	11.5 (5.4)	9.0 (7.5)	10.9 (5.2)											
Effect size T ₀ T ₁	0.3	0.2	0.4	0.2	0.1	0.5	0.3											
CABAH 4	(N = 82)	(N = 30)	(N = 11)	(N = 12)	(N = 11)	(N = 10)	(N = 8)											
T ₀	7.2 (4.1)	6.6 (4.2)	7.6 (3.3)	8.8 (4.0)	9.3 (4.7)	5.5 (3.8)	5.4 (3.2)											
T ₁	7.3 (3.9)	6.8 (4.5)	7.2 (2.8)	9.3 (3.5)	8.8 (4.9)	5.9 (2.5)	5.5 (2.3)											
T ₂	6.5 (3.9)	6.3 (4.1)	6.0 (3.4)	7.8 (4.3)	8.0 (4.2)	6.1 (3.7)	4.0 (2.7)											
T ₃	5.7 (3.9)	5.6 (4.0)	6.4 (4.0)	7.5 (4.7)	6.4 (3.3)	3.7 (3.2)	4.3 (3.4)											
Effect size T ₀ T ₁	0.4	0.2	0.3	0.3	0.7	0.4	0.3											
CABAH 5	(N = 86)	(N = 30)	(N = 16)	(N = 9)	(N = 12)	(N = 12)	(N = 7)											
T ₀	7.1 (2.4)	7.3 (2.0)	7.0 (2.2)	6.9 (1.8)	7.8 (2.8)	6.4 (3.4)	7.1 (3.2)											
T ₁	6.9 (2.4)	7.2 (2.2)	6.9 (1.7)	7.0 (1.4)	7.7 (2.6)	5.5 (2.7)	6.7 (3.7)											
T ₂	7.2 (2.4)	7.6 (2.0)	7.9 (2.3)	6.8 (2.1)	7.7 (2.6)	5.8 (2.6)	6.7 (3.6)											
T ₃	6.8 (2.2)	6.9 (2.2)	7.1 (1.6)	7.0 (2.2)	6.6 (2.9)	6.4 (2.2)	6.3 (2.8)											
Effect size T ₀ T ₁	0.1	0.2	0.0	0.0	0.6	0.0	0.4											

*p < .05; **p < .01; ***p < .001.

the effects of the different types of axis II comorbidity, a slight but nonsignificant tendency toward a poorer outcome of subjects with multiple cluster C PDs was found on somatoform symptom and hypochondriasis indices. One reason for the absence of significant differences could be that our sample size was rather small for the assessment of six groups although it is large compared with other studies on the effects of comorbid PDs.

Conclusions

Our results are consistent with the recent findings on cognitive behavioral treatment outcome of anxiety, depressive, and obsessive-compulsive disorders (Dreessen et al., 1994, 1997; Shea et al., 1990) that found no distinct effects of comorbid PDs. However, the total number of studies that investigated the influence of comorbid PDs on psychotherapeutic treatment is very small until now. One important issue of this study is the fact that markedly more subjects with than without PDs (regardless of their axis I diagnoses) dropped out, which was also reported in a study by Persons et al. (1988) on depression. The higher drop-out rate might indicate a poorer treatment outcome. On the other hand, it could be an expression of well-known interactional problems in PDs, which need not necessarily affect treatment outcome of axis I disorders. Further studies with larger samples and follow-up assessments are required to determine differential effects of axis II pathology as well as long-term effects of comorbid PDs, which are commonly characterized as very persistent disorders with high relapse rates.

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